Echocardiography in stroke and transient ischaemic attack

John B Chambers, Mark A de Belder, David Moore

Stroke and transient ischaemic attack (TIA) remain controversial indications for echocardiography. In some centres they represent 40–50% of the total requests for transoesophageal studies, while many stroke units never request echocardiography as a matter of routine. We need to examine the reasons for these wide differences in practice.

How is cardiac stroke diagnosed?
Approximately 20% of strokes are caused by cerebral haemorrhage and 80% by infarction.1 Of the infarctions, a proportion are thrombotic and the rest as a result of emboli either from artery to artery within the cerebral circulation or from the heart or aorta. The diagnosis of a cardiac source for emboli is based to some degree on the clinical presentation and the results of computed tomography or magnetic resonance imaging (table 1),2 but largely on the findings on echocardiography itself. This has introduced a bias that has tended to overstate the importance of echocardiography particularly via the transoesophageal approach. Thus, studies based on transthoracic echocardiography have suggested that approximately 15% of TIAs and 15–35% of cerebral infarcts are cardiac in origin1 while transoesophageal studies suggest figures of at least 50% and up to 83%.4 Some echocardiographic abnormalities, however, are controversial or even spurious causes of stroke—for example, mitral prolapse, atrial septal aneurysm or mitral annular calcification. The role of echocardiography in patients suspected of suffering a cardiac embolus must therefore be examined critically.

Role of echocardiography in patients with stroke
Echocardiography may find a direct source of emboli—for example, thrombus, myxoma or vegetation (table 2). Up to 1% of all strokes in patients aged under 50 years may be caused by a left atrial myxoma1 and up to 15% of patients with endocarditis present with an embolic event.6 However, more usually, echocardiography identifies a condition with a known risk of emboli—for example, mitral stenosis may be the underlying cause of cerebral infarction even if a thrombus cannot be imaged. The thrombus might be beyond the resolution of the technique or might already have embolised. Another example is dilated cardiomyopathy where the risk of stroke is 5% per year regardless of whether left ventricular thrombus is identified on echocardiography.5

When should transthoracic echocardiography be requested?
In the absence of a significant abnormality on clinical cardiovascular examination or on the electrocardiogram, the yield from transthoracic echocardiography is virtually zero and it should not be performed routinely.8,9,12 However, in young subjects (younger than 50), transthoracic examination is usually done as the immediate prelude to transoesophageal echocardiography. Some authors have suggested that the transthoracic study can guide the use of transoesophageal echocardiography. Thus, in two studies the presence of 92%13 and 95%14 of abnormalities on transoesophageal examination were predicted by transthoracic abnormalities such as left atrial enlargement, left ventricular hypertrophy or mitral valve thickening. These studies suggested that transoesophageal echocardiography was not necessary in patients aged under 40 years with a normal transthoracic study. However, at least anecdotally, there is still a small yield of unsuspected vegetations or thrombi on transoesophageal examination.

It could be argued that if a significant clinical abnormality such as atrial fibrillation or mitral stenosis is found, echocardiography is no longer necessary. However, echocardiography will still refine the risk of embolisation. For example, in a patient in atrial fibrillation the risk of stroke is low if the echocardiogram is completely normal, but progressively higher if...
the left atrium is dilated and the left ventricle hypokinetic (table 3). In a patient with mitral stenosis, echocardiography may reveal a ball thrombus for which the management becomes urgent surgery rather than simply anticoagulation.

Atrial fibrillation is a marker for stroke rather than necessarily the direct cause. It is associated with other conditions such as hypertension or heart failure, which themselves carry an independent risk of stroke. For this reason atrial fibrillation is found in 11% of patients suffering cerebral haemorrhage as well as 25–35% of those with cerebral thrombosis. These figures are lower than the incidence of 60% in patients with embolism, but they still underline that brain imaging to exclude haemorrhage should always be performed in patients in atrial fibrillation before starting anticoagulation.

How important is transoesophageal echocardiography?

In contrast to transthoracic echocardiography, the transoesophageal approach frequently shows abnormalities even in the absence of clinical signs (table 4). These include patent foramen ovale, atrial septal aneurysm, left atrial spontaneous contrast, left atrial thrombus, and aortic atheroma. However, the importance of many of these findings is difficult to evaluate because of a lack of standardisation of definitions and a relative lack of control comparisons.

Table 4 Possible causes of stroke found on echocardiography

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<th>Dilated left ventricle</th>
<th>Hypertrophic cardiomyopathy (with atrial fibrillation)</th>
<th>Restrictive myopathy</th>
<th>Endomyocardial fibrosis</th>
<th>Thrombus (lupus, malignancy, thromboembolia, polycystic kidney disease)</th>
<th>Ventricular septal defect with pulmonary hypertension</th>
<th>Valve</th>
<th>Mitral stenosis</th>
<th>Replacement heart valve (aortic or mitral)</th>
<th>Endocarditis</th>
<th>Noninfective endocarditis (eg, in malignancy)</th>
<th>Fibroembolism</th>
<th>Left atrium</th>
<th>Large left atrium/left atrial spontaneous contrast</th>
<th>Thrombus</th>
<th>Atrial septal aneurysm</th>
<th>Patent foramen ovale</th>
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Table 6  Left atrial spontaneous contrast and stroke

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Table 7  Left atrial thrombus and stroke

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Table 8  Atrial septal aneurysm and stroke

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NS, not stated.

Table 9  Aortic atheroma and stroke

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LEFT ATRIAL SPONTANEOUS CONTRAST AND THROMBUS

Left atrial spontaneous contrast is shown in about 20% of patients after stroke compared with 5% of controls (table 6). Thrombus is found in 10% with stroke compared with 3% of controls (table 7) and may coexist with spontaneous contrast and may occur separately. As spontaneous contrast varies with gain settings and the carrying frequency of the probe, it is a relatively non-specific finding and future work must aim at grading the density of the effect.

ATRIAL SEPTAL ANEURYSM

Significant problems exist with the definition of atrial septal aneurysm. The threshold excursion ranges from as little as 6 mm to as much as 15 mm which must largely explain the wide variation in incidence between 1% and 28% (table 8). However, all studies agree that approximately three-quarters of aneurysms contain a patent foramen ovale. This is the most likely cause of stroke in such patients although there is rare anecdotal evidence of thrombus forming at the apex of an aneurysm.

AORTIC ATEROMA

Again there are problems with definitions of aortic atheroma. Some authors report any degree of aortic atheroma giving an incidence of 22% v 4% in controls (table 9). There is good agreement that only large or complex atheroma constitute a significant risk of stroke with an adjusted relative risk of 7.1. This compares with a risk of 2.3 with simple atheroma and 2.1 with other intracardiac sources of emboli.

KARALIS ET AL. found that embolic events occurred in 61% of patients with atheroma compared with only 4% in control subjects. Embolism was especially likely, in 73% of cases, when there was mobile atheroma protruding more than 5 mm into the lumen. This compared with an incidence of 12% in patients with simple atheroma. The risk of embolism was substantially increased with invasive interventions and occurred in four of 15 patients (27%) with complex atheroma undergoing cardiac catheterisation or placement of aortic balloon pumps.

CONTROVERSIAL CAUSES OF STROKE

Some abnormalities may not genuinely be related to stroke. For example, although mitral prolapse has been reported in up to 40% of patients with ischaemic stroke, in most studies it is far less common and in some there were no cases at all. In the large SPAF study the incidence was 5%, the same as in the control group. Furthermore the incidence of stroke in patients with mitral prolapse is low and can be accounted for by other associated factors such as atrial fibrillation.

MITRAL ANNULAR CALCIFICATION may be related to stroke although the cause is not clear. In the Boston trial of warfarin in non-rheumatic atrial fibrillation, mitral annular calcification was present in 67% of patients with stroke compared with 29% without. A link between mitral annular calcification and aortic atheroma has been postulated although never tested, and at present we believe that there is insufficient echocardiographic evidence to assume that mitral annular calcification is an independent risk factor for stroke, nor is it clear how finding mitral annular calcification should alter management.

Does transoesophageal echocardiography affect management?

Many studies overemphasise the role of echocardiography in stroke by reporting even mild degrees of valve thickening or mitral prolapse leading to a rate of abnormality as high as 83%. Many of these patients have other causes of stroke such as atrial fibrillation or carotid atheroma, and a comprehensive assessment of the patient must be made rather than relying on the echocardiogram alone. Even then, about 30% of patients will have a potentially important abnormality on transoesophageal echocardiography.

Do these findings change management? In most cases where left atrial spontaneous contrast or thrombus are found the patient is in atrial fibrillation so will already have been anticoagulated with warfarin (in the absence of contraindications). If the risk of haemorrhage has been judged too high to start warfarin, the
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Table 10 Indications for echocardiography in patients with stroke or TIA

<table>
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<tr>
<th>Procedure</th>
<th>Age under 50 years</th>
<th>Age under 50 years</th>
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<td>Transfemoral</td>
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Table 11 Recommendations for performing transoesophageal echocardiography

1. Look at the left atrial appendage for thrombus and spontaneous contrast
2. Check for spontaneous contrast
3. Perform a contrast study. Use 10 ml dextrose saline or saline with 1 ml air and 1 ml of the patient's blood shot between two syringes using a three way tap and injected at speed on three occasions using different views of the atrial septal aneurysm.
4. Assess the aorta for the aortic root.
5. Inspect the left atrium for spontaneous contrast.
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When should we recommend echocardiography?

Transesophageal echocardiography on its own is only clearly indicated in patients aged over 50 years where there is a relevant abnormality on clinical examination or the electrocardiogram. Transesophageal echocardiography together with transthoracic examination is indicated in younger subjects to look for rare treatable causes such as myxoma or endocarditis, or the occasional thrombus found in the absence of atrial fibrillation or mitral stenosis (table 10).14 Computed tomography or magnetic resonance imaging must always be performed first as even atrial fibrillation or left atrial thrombus can be associated with cerebral haemorrhage.

There is an argument for subjecting all patients to transesophageal echocardiography if neurological and haematological investigations are unrewarding. The incidence of patent foramen ovale in transoesophageal echocardiography examination is higher in the elderly than in the young.42 This is the reverse of the pattern seen at postmortem43 and may be explained by the fact that patent foramina in the elderly tend to be larger than in the young43 and therefore more readily detectable. The elderly are also more likely to have deep vein thrombosis or pulmonary hypertension. It is therefore possible that patent formen ovale is equal or more important in this age group. Transesophageal echocardiography might be useful for stratifying risk to help decide who should have anticoagulation. However, no evidence exists to support this logical approach. We need a prospective study to see whether transoesophageal echocardiography can predict further events and whether a high risk group that will benefit from warfarin can be identified. Current recommendations for performing echocardiography are given in table 11.


38 Mas JL, Zubera M. Recurrent cerebrovascular events in patients with patent foramen ovale, atrial septal aneurysm or both and cryptogenic stroke or transient ischaemic attack. Am Heart J 1995;130:1083-8.


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*Heart* 1997 78: 2-6
doi: 10.1136/hrt.78.Suppl_1.2